OBSTETRICS

Trends in postpartum hemorrhage: United States, 1994–2006

William M. Callaghan, MD, MPH; Elena V. Kuklina, MD, PhD; Cynthia J. Berg, MD, MPH

OBJECTIVE: The purpose of this study was to estimate the incidence of postpartum hemorrhage (PPH) in the United States and to assess trends.

STUDY DESIGN: Population-based data from the 1994–2006 National Inpatient Sample were used to identify women who were hospitalized with postpartum hemorrhage. Data for each year were plotted, and trends were assessed. Multivariable logistic regression was used in an attempt to explain the difference in PPH incidence between 1994 and 2006.

RESULTS: PPH increased 26% between 1994 and 2006 from 2.3% (n = 85,954) to 2.9% (n = 124,708; $P < .001$). The increase primarily was due to an increase in uterine atony, from 1.6% (n = 58,597) to 2.4% (n = 99,904; $P < .001$). The increase in PPH could not be explained by changes in rates of cesarean delivery, vaginal birth after cesarean delivery, maternal age, multiple birth, hypertension, or diabetes mellitus.

CONCLUSION: Population-based surveillance data signal an apparent increase in PPH caused by uterine atony. More nuanced clinical data are needed to understand the factors that are associated with this trend.

Key words: postpartum hemorrhage, pregnancy, uterine atony

Cite this article as: Callaghan WM, Kuklina EV, Berg CJ. Trends in postpartum hemorrhage: United States, 1994–2006. Am J Obstet Gynecol 2010;202:353.e1-6.

Postpartum hemorrhage (PPH) is a frequent complication of pregnancy and is among the most common causes of pregnancy-related death in the United States.[1] Recent reports demonstrated a rising trend in severe maternal morbidity during US delivery hospitalizations

★ EDITORS' CHOICE ★

that was attributable largely to the increased use of blood transfusions.[2,3] In addition, data from Canada and Australia indicate recent increases in PPH rates[4,5] and aggregate US data show that the percentage of women whose discharge records contained *International Classification of Diseases, 9th Revision, Clinical Modification* (*ICD-9-CM*) codes for PPH increased from 2.0% in 1993–1997 to 2.6% in 2001–2005.[6]

PPH is an etiologically heterogeneous event and not a diagnosis. The causes of PPH include poor uterine tone (uterine atony), retained placental tissue, abnormalities of placentation, genital tract trauma, and abnormalities of coagulation. A recent report documented that apparent increases in rates of PPH largely were due to an increase in the use of the *ICD-9-CM* code for uterine atony.[4] As a first step to better understand the problem of PPH in the United States, we undertook a descriptive analysis of US population-based administrative hospital discharge data to examine trends in PPH, with special attention to the contribution of uterine atony and associated obstetric factors.

Materials and Methods

Data for this investigation were obtained from the Nationwide Inpatient Sample (NIS) for the years 1994–2006. The NIS is part of the Agency for Healthcare Research and Quality's Healthcare Cost and Utilization Project. The details of the NIS have been described in detail elsewhere.[7] Briefly, the NIS is the largest inpatient care database in the United States; after appropriate weighting, NIS data are intended to be representative of all patients who are admitted to US hospitals. During annual data collection by the Healthcare Cost and Utilization Project, all nonfederal community hospitals from participating states are stratified by rural/urban location, number of beds, region of the country, teaching status, and ownership. Within each stratum, a systematic random 20% sample of hospitals is drawn. The database contains ≤15 diagnosis fields and 15 procedure fields for each discharge; diagnoses and procedures are coded at the hospital at discharge with the *ICD-9-CM* codes. Because the NIS is available to the public and does not contain any personal identifying information, this investigation did not require approval by an institutional review board.

Except for age, the NIS does not collect individual demographic information, nor does it report obstetric characteristics for individual pregnancies, except those that can be translated to *ICD-9-CM* codes. As such, this analysis focuses on *ICD-9-CM* diagnosis codes for obstetric hemorrhage and coded

From the Divisions of Reproductive Health (Drs Callaghan and Berg) and Heart Disease and Stroke Prevention (Dr Kuklina), Centers for Disease Control and Prevention, Atlanta, GA.

Presented in part at a meeting of the International Postpartum Haemorrhage Collaborative Group in Montreal, QC, Canada, Nov. 10, 2008.

Received July 24, 2009; revised Oct. 16, 2009; accepted Jan. 12, 2010.

Reprints: William M. Callaghan, MD, MPH, Division of Reproductive Health, Centers for Disease Control and Prevention, 4770 Buford Hwy, MS K-23, Atlanta, GA 30341. wcallaghan@cdc.gov.

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

Authorship and contribution to the article is limited to the 3 authors indicated. There was no outside funding or technical assistance with the production of this article.

0002-9378/free
Published by Mosby, Inc.
doi: 10.1016/j.ajog.2010.01.011

For Editors' Commentary, see Table of Contents

TABLE 1
Maternal and hospitalization characteristics, 1994 and 2006

Characteristic	Percentage ± SE[a] 1994 (n = 3,791,724)	2006 (n = 4,260,198)
Age, y		
<20	12.8 ± 0.4	10.5 ± 0.3
20-34	75.1 ± 0.2	75.4 ± 0.2
>34	12.1 ± 0.3	14.2 ± 0.4
Payer		
Medicaid/Medicare	38.7 ± 1.3	42.9 ± 1.3
Private insurance	53.0 ± 1.5	50.3 ± 1.6
Self	8.3 ± 0.8	6.8 ± 0.7
Mode of delivery		
Vaginal	74.8 ± 0.3	67.0 ± 0.3
Vaginal birth after cesarean	4.0 ± 0.1	1.5 ± 0.1
Repeat cesarean	7.7 ± 0.1	13.5 ± 0.2
Primary cesarean	13.4 ± 0.2	18.1 ± 0.3
Labor induction	9.8 ± 0.4	16.3 ± 0.5
Multiple birth	1.4 ± 0.0	1.7 ± 0.0
Hypertension[b]	5.6 ± 0.1	8.1 ± 0.1
Diabetes mellitus[b]	3.4 ± 0.1	6.1 ± 0.1

Probability values are all < .001 for comparisons between 1994 and 2006.

[a] May not total to 100 because of rounding; [b] Includes gestational and pregestational conditions.

Callaghan. Trends in postpartum hemorrhage. Am J Obstet Gynecol 2010.

characteristics of the delivery. Following the validated methods of Kuklina et al,[8] we characterized delivery hospitalizations using a hierarchic algorithm based on *ICD-9-CM* diagnosis and procedure codes and diagnosis-related group codes.

There are 4 *ICD-9-CM* codes for PPH. Cases of uterine atony at delivery were identified by the 5-digit code 666.1X. All other PPH was identified by the PPH codes 666.0X (retained, trapped, or adherent placenta), 666.2X (delayed and secondary PPH), and 666.3X (postpartum coagulation defects). We collapsed the latter 3 codes (for cases of PPH not attributable to uterine atony) into a single category labeled "other hemorrhage." Cesarean section delivery was identified by *ICD-9-CM* code 74.X, induction by 73.4, previous cesarean section delivery by 654.2X, and blood transfusions by 99.03 and 99.04.

We calculated rates as percentages of deliveries, plotted annual rates from 1994-2006, and assessed the significance of trends in rates by calculating orthogonal polynomial contrasts according to the methods of Fisher and Yates as described in the *SUDAAN Example Manual*.[9] In an attempt to explain trends in rates, we used logistic regression to model uterine atony as a function of time, maternal age, induced labor, cesarean delivery, multiple birth, hypertension during pregnancy, diabetes mellitus during pregnancy, and hospital location and characteristics. We compared rates of uterine atony in 2006 with those in 1994 for each mode of delivery after age standardizing the 2006 rate to the age distribution of women who delivered in 1994. Finally, the odds of atony for each mode of delivery were calculated for 1994 and for 2006 with adjustment for age. All counts and proportions were weighted with the use of the weighting variables in the NIS that account for the complex sampling design. Hence, estimates are generalizable to the US population. All analyses were performed with SAS software (version 9.1; SAS Institute Inc, Cary, NC) and SAS-callable SUDAAN (version 9.0; RTI International, Research Triangle, NC).

RESULTS

From 1994–2006, the NIS collected data on 10,481,197 delivery hospitalizations, which represented a weighted estimate of 51,674,542 delivery hospitalizations in the United States during that period; 2.7% of the women who were discharged after delivery during that period received a code for PPH. Three-fourths of these women were identified by the presence of the single code for uterine atony. Two of every 1000 women with a PPH code also had a code for blood transfusion at discharge. One in 4 women delivered by cesarean section, and 1 in 7 women had labor induction. Maternal characteristics of the population and the characteristics of the delivery hospitalizations were different between 1994 and 2006 (Table 1). In 2006, women were older and more likely to use government insurance; they were more likely to deliver by cesarean section or after induction of labor, have a multiple gestation, and have pregnancies that were complicated by hypertension and diabetes mellitus. In 2006, women were less likely to have a vaginal birth when a previous birth occurred by cesarean section delivery.

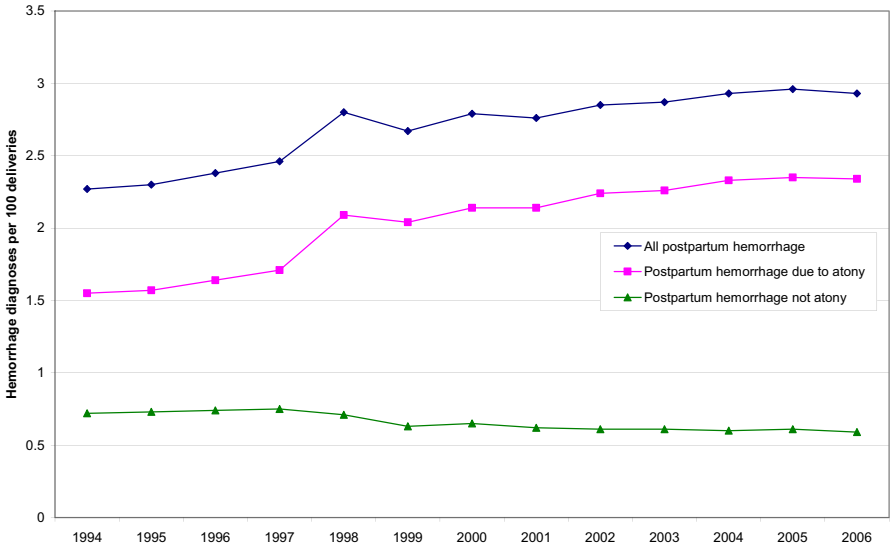
Between 1994 and 2006, the percentage of deliveries with a code for PPH increased by 26%, from 2.3% (85,954 deliveries) to 2.9% (124,708 deliveries; test of trend, $P < .001$; Figure 1). There was a parallel increase in PPH caused by atony during this same time period, from 1.6% (58,597 cases) to 2.4% (99,904 cases; $P < .001$). Delivery hospitalizations with PPH codes not caused by atony did not increase ($P > .05$). Multivariable logistic regression with simultaneous adjustment for all variables that are given in Table 1 and hospital size, urban vs rural location, geographic region, and teaching status showed no significant effect of these variables on the change in the risk of PPH between 1994 and 2006.

The percentage of delivery hospitalizations with the *ICD-9-CM* code for uterine atony varied by the mode of delivery

and whether pregnancy was induced (Figure 2). The highest rate of uterine atony occurred among women whose labor was induced and who delivered vaginally. This was followed by women whose induction ended in cesarean delivery and women who had vaginal births without induction of labor. Women who had cesarean deliveries and did not have induced labor consistently had the lowest rates of PPH caused by atony. The percentage of women with a PPH code that indicated atony who also had a code for blood transfusion more than doubled between 1994 and 2006 (test of trend, $P < .001$; Figure 3).

In 1994, the group at lowest risk of PPH from atony was women who delivered by cesarean section with no induction (Table 2). Compared with that group, women who had vaginal deliveries with induced labor had the highest risk of atonic PPH (odds ratio [OR], 3.7), followed by women who had vaginal deliveries without induced labor (OR, 2.5) and women who had cesarean deliveries after induced labor (OR, 1.8). Between 1994 and 2006, the overall rate of PPH caused by uterine atony increased by 50%. This rate increased among women with all 4 combinations of delivery method and induction status (test for trend, $P < .001$; Figure 2). However, the percent increase differed according to induction status and method of delivery (Table 2). For both induced and noninduced labors, the increase in atony rates among women who had cesarean deliveries was greater than the increase among women who had vaginal deliveries. In fact, for the last 2 years of observation, the rates of uterine atony for women whose labor was induced were virtually identical for women with vaginal or with cesarean delivery (Figure 2). By 2006, the rates of hemorrhage and the ORs for PPH caused by uterine atony were more similar than the rates and ORs in 1994 for both modes of delivery regardless of labor induction. The percentage change in uterine atony was dramatically greater for cesarean deliveries compared with vaginal deliveries, which resulted in decreased ORs for vaginal deliveries in 2006 when noninduced cesarean deliveries served as the reference group (Table 2).

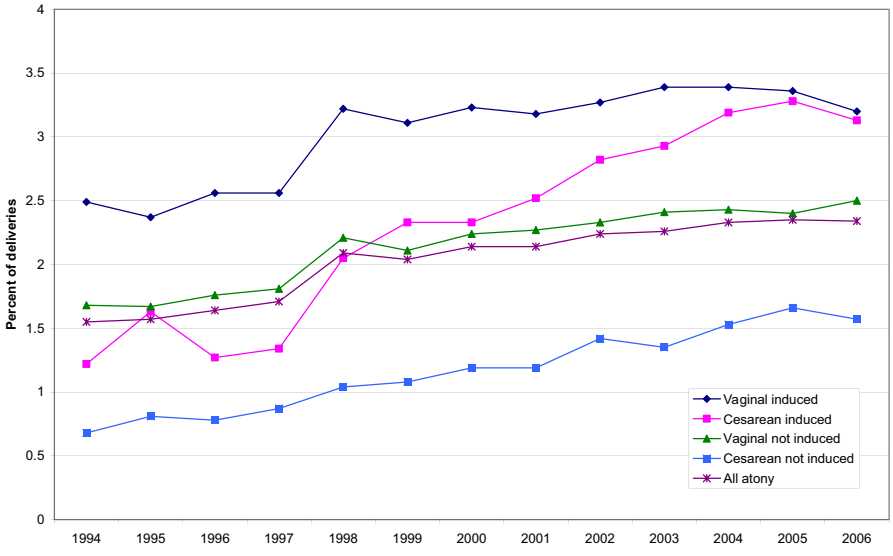
FIGURE 1
Annual postpartum hemorrhage rates (United States, 1994–2006)



Callaghan. Trends in postpartum hemorrhage. Am J Obstet Gynecol 2010.

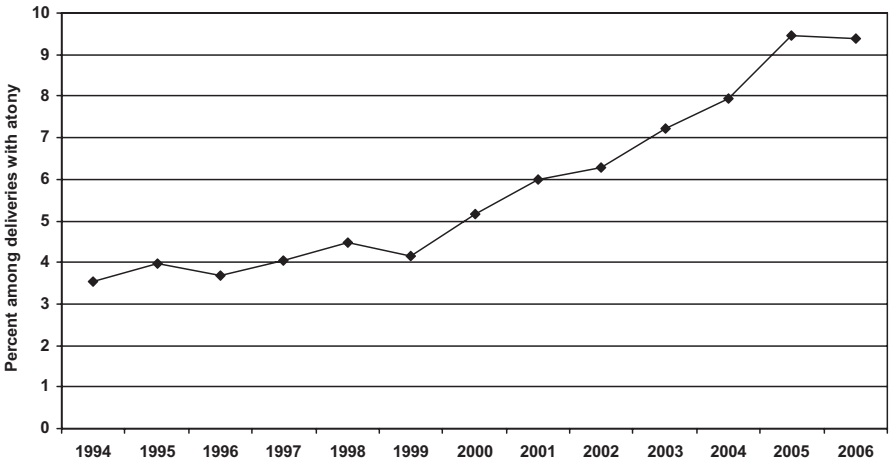
The distribution of deliveries in 1994 and 2006 by the use of labor induction and delivery method is shown in Table 3. When these changes in the distribution of labor induction and methods of delivery were taken into account, the proportion of all cases of uterine atony that occurred among women who had a vaginal delivery decreased from 90–77%; the proportion among women who delivered by cesarean section increased from 10-23%. Because of an increase in the percentage of women who had cesarean deliveries, women who delivered by cesarean section without labor induction (the group with the lowest rate of uterine

FIGURE 2
Annual rates of postpartum hemorrhage caused by atony, by mode of delivery, and by induction status (United States, 1994–2006)



Callaghan. Trends in postpartum hemorrhage. Am J Obstet Gynecol 2010.

FIGURE 3
Transfusion rates among women with postpartum hemorrhage caused by atony (United States, 1994–2006)



Callaghan. Trends in postpartum hemorrhage. Am J Obstet Gynecol 2010.

atony) made the greatest contribution to the increased number of uterine atony cases between 1994 and 2006.

Comment

The percentage of US women whose hospital discharge records after a delivery contained *ICD-9-CM* codes for PPH increased substantially between 1994 and 2006. This increase was driven primarily by an increase in the percentage of women with an *ICD-9-CM* code for uterine atony, and it occurred for vaginal and cesarean deliveries regardless of induction status. The accompanying disproportionate increase in transfusions suggests the possibility of an increase in severity of PPH caused by atony, although there was some evidence of a general increase in the use of transfusion across the United States.[10] Interestingly, maternal mortality rates for deaths caused by childbirth-associated hemorrhage did not change substantially over this same time period.[11,12]

Administrative discharge data such as those from the NIS are particularly useful for population-based surveillance. Although lacking in clinical detail, large hospitalization databases can suggest that events or conditions are increasing or decreasing in frequency or severity. Although the increase that we found in the reported rate of PPH caused by uterine atony could have alternative explanations (such as changes in coding practices, changing definitions of the event, or a general increased awareness and documentation of the event), our findings are consistent with other recently published reports. Over a similar time period and with the use of similar methods, Joseph et al[4] reported an increase in PPH from 4.1-5.1% in Canada that largely was due to an increase in the rate of uterine atony; adjustments for changes in the prevalence of PPH risk factors did not explain the increase. A similar increase in PPH rates from 1994 through 2002 was reported in New South Wales, Australia.[5]

The trend in the prevalence of PPH in large databases may be affected by the definition of the event over time. Although there is no evidence that definitions have changed in recent years, it is important to note that several definitions have been used. A traditional definition for PPH in the United States is an estimated blood loss of at least 500 mL for a vaginal delivery and 1000 mL for a cesarean delivery.[13] These criteria are based on mean blood losses from 75 women who delivered vaginally and 40 women who delivered by cesarean section; nearly one-half of these women lost in excess of these limits.[14] In contrast, an online coding manual defines PPH as blood loss in excess of 500 mL without

TABLE 2
Estimated rates[a] of uterine atony and corresponding odds ratios, 1994 and 2006

	Rate (95% CI)			Odds ratio (95% CI)	
Delivery type	**1994**	**2006**	**Change, %**	**1994**	**2006**
Overall	1.6 (1.4–1.8)	2.4 (2.2–2.5)	50		
Vaginal					
Induced	2.5 (2.3–2.8)	3.2 (3.0–3.5)	28	3.7 (3.2–4.4)	2.1 (1.9–2.2)
Not induced	1.7 (1.5–1.9)	2.5 (2.3–2.7)	47	2.5 (2.2–2.9)	1.6 (1.5–1.7)
Cesarean					
Induced	1.2 (0.9–1.6)	3.1 (2.8–3.5)	160	1.8 (1.4–2.3)	2.0 (1.8–2.2)
Not induced	0.7 (0.6–0.8)	1.6 (1.5–1.8)	130	Reference	Reference

CI, confidence interval.

[a] Age-adjusted to the age distribution of the 1994 delivery hospitalization population.

Callaghan. Trends in postpartum hemorrhage. Am J Obstet Gynecol 2010.

TABLE 3
Distribution of deliveries and number of uterine atony cases, 1994 and 2006

	Delivery distribution, %[a]		Cases of postpartum hemorrhage caused by uterine atony, n[a]		
Type of delivery	1994	2006	1994	2006	Overall increase in cases
Overall	100	100	58,597	99,905	41,308
Vaginal	78.8	68.9	52,815 (90.2%)	76,760 (76.8%)	23,945 (58.0%)
Induced	8.1	13.2	7651 (13.1%)	17,972 (18.0%)	10,321 (25.0%)
Not induced	70.7	55.7	45,164 (77.1%)	58,788 (58.8%)	13,624 (33.0%)
Cesarean	21.2	31.1	5782 (9.9%)	23,145 (23.2%)	17,363 (42.0%)
Induced	1.7	3.1	790 (1.3%)	4127 (4.1%)	3337 (8.1%)
Not induced	19.5	28.0	4992 (8.5%)	19,018 (19.0%)	14,026 (34.0%)

[a] Percentages may not total to 100 because of rounding.

Callaghan. Trends in postpartum hemorrhage. Am J Obstet Gynecol 2010.

reference to mode of delivery.[15] However, it is not apparent that the physiologic effect of blood loss differs by mode of delivery; blood loss of ≥1000 mL is associated with considerable morbidity, compared with lesser amounts.[16] Moreover, estimating blood loss is fraught with error; visual estimation consistently underestimates blood loss compared with calculated estimates that are based on estimated maternal blood volume and changes in hematocrit level before and after delivery.[17] It is unknown how variation in the definition of PPH or how blood loss is estimated affects variation in coding or how medical coders use narrative in the medical record, checkboxes in labor and delivery summaries, and written estimates to arrive at a coded diagnosis for uterine atony. However, because cesarean deliveries may have to meet a higher standard for blood loss to meet the definition of PPH compared with vaginal deliveries, our finding that noninduced cesarean deliveries had the lowest PPH rate might be explained partially by the difference in case definition of PPH for these modes of delivery.

Prevention of PPH remains a challenge. Risk factors for PPH, which include high infant birthweight, induced labor, chorioamnionitis, and multiple gestations, are well-established.[16,18] However, these risk factors have only moderate positive predictive value for PPH caused by uterine atony and hence have limited clinical value. Although the active management of the third stage of labor has been demonstrated to reduce the occurrence of PPH,[19] a recent Australian report documented significant variation in practice regarding the management of the third stage of labor.[20] The extent to which active management occurs in the United States is not known. However, a multifaceted behavioral intervention has been shown to be effective in increasing the rates of prophylactic oxytocin use during the third stage of labor.[21] Secondary prevention of hemorrhage relies on the accurate and timely estimation of blood loss in real time, and researchers who use simulation exercises have reported improvements in estimates of maternal blood loss during delivery.[22,23]

Our study was limited by its reliance on administrative hospital discharge data. Although morbidity codes tend to be specific and have good negative predictive values, they often are not sensitive.[24-26] Hence, our use of coded discharge data likely caused us to underestimate the incidence of PPH. Until a uniform definition of clinically meaningful PPH is widely accepted and tools for the assessment of blood loss at delivery are widely adopted, it will be difficult to establish a medical record gold standard for use in validation studies. It is also possible that cases of hemorrhage that was not caused by atony (such as abnormalities of placentation [*ICD-9-CM* 666.0X]) are incorrectly coded as atony. We found that labor induction is an important risk for PPH caused by atony. However, to the extent that our data lacked sensitivity to identify induced labor, we may have underestimated the true risk. Last and perhaps most important, administrative discharge data have minimal information about maternal and infant characteristics (such as obesity and birthweight) and events that occurred during labor and delivery that might have contributed to the risk of uterine atony. Specifically, although the *ICD-9-CM* includes procedure codes for induction, it does not include a specific code to indicate whether labor occurred; as a result, we could not determine reliably whether labor preceded noninduced cesarean deliveries. Similarly, because of the likelihood of confounding or interaction by labor status, we were unable to perform a meaningful assessment of the impact of primary and repeat cesarean section delivery on PPH. The apparent leveling of the increase in PPH in the most recent years is coincident with the general increase in cesarean deliveries; the inability to understand the relationships among primary and repeat cesarean deliveries and procedures that were performed without or after labor limits our ability to explain our observations with these data.

Taken together with similar reports from other countries with well-developed systems for delivery of health services, the apparent increase in PPH in recent years suggests an emerging threat to women during labor and delivery. Despite limitations, when these data are

viewed from the perspective of surveillance and not from the perspective of etiologic research, the increasing trend in uterine atony that we present might be viewed as a signal that requires attention and investigation. Ideally, confirmatory investigations could be carried out in large delivery hospitals or hospital systems with access to more nuanced data about pregnant women and their delivery experiences. ■

REFERENCES

1. Berg CJ, Chang J, Callaghan WM, Whitehead SJ. Pregnancy-related mortality in the United States, 1991-1997. Obstet Gynecol 2003;101:289-96.

2. Callaghan WM, MacKay AP, Berg CJ. Identification of severe maternal morbidity during delivery hospitalizations, United States, 1991-2003. Am J Obstet Gynecol 2008;199:133.e1-8.

3. Kuklina EV, Meikle SF, Jamieson DJ, et al. Severe obstetric morbidity in the United States: 1998-2005. Obstet Gynecol 2009;113:293-9.

4. Joseph KS, Rouleau J, Kramer MS, Young DC, Liston RM, Baskett TF. Investigation of an increase in postpartum haemorrhage in Canada. BJOG 2007;114:751-9.

5. Ford JB, Roberts CL, Simpson JM, Vaughan J, Cameron CA. Increased postpartum hemorrhage rates in Australia. Int J Gynecol Obstet 2007;98:237-43.

6. Berg CJ, MacKay AP, Qin C, Callaghan WM. Overview of maternal morbidity during hospitalization for labor and delivery, United States: 1993-1997 and 2001-2005. Obstet Gynecol 2009;113:1075-81.

7. Healthcare Cost and Utilization Project HCUP. Overview of the Nationwide Inpatient Sample, (NIS). Available at: www.hcup-us.ahrq.gov/databases.jsp. Accessed Feb. 26, 2009.

8. Kuklina EV, Whiteman MK, Hillis SD, et al. An enhanced method for identifying obstetric deliveries: implications for estimating maternal morbidity. Matern Child Health J 2008;12:469-77.

9. SUDAAN example manual. Research Triangle Park, NC: Research Triangle Institute; 2004.

10. Sullivan MT, Cotton R, Read EJ, Wallace EL. Blood collection and transfusion in the United States in 2001. Transfusion 2007;47:385-94.

11. Singh GK, Kochanek KD, MacDorman MF. Advance report of final mortality statistics, 1994: monthly vital statistics report; vol 45, no. 3, supp. Hyattsville, MD: National Center for Health Statistics; 1996.

12. Kung HC, Hoyert DL, Xu JQ, Murphy SL. Deaths: final data for 2005: national vital statistics reports; vol 56, no. 10. Hyattsville, MD: National Center for Health Statistics; 2008.

13. American College of Obstetricians and Gynecologists (ACOG). Postpartum hemorrhage: ACOG practice bulletin no. 76. Obstet Gynecol 2006;108:1039-47.

14. Pritchard JA, Baldwin RM, Dickey JC, Wiggins KM. Blood volume changes in pregnancy and the puerperium. Am J Obstet Gynecol 1962;84:1271-82.

15. Ingenix Encoder Pro.com Professional. Available at: http://www.encoderpro.com/epro/. Accessed Feb. 23, 2009.

16. Lu MC, Korst LM, Fridman M, Muthengi E, Gregory KD. Identifying women most likely to benefit from prevention strategies for postpartum hemorrhage. J Perinatol 2009. Epub ahead of print.

17. Stafford I, Dildy GA, Clark SL, Belfort MA. Visually estimated and calculated blood loss in vaginal and cesarean delivery. Am J Obstet Gynecol 2008;199:519.e1-7.

18. Rouse DJ, Leindecker S, Landon M, et al. The MFMU Cesarean Registry: uterine atony after primary cesarean delivery. Am J Obstet Gynecol 2005;193:1056-60.

19. Prendiville WJP, Elbourne D, McDonald SJ. Active versus expectant management in the third stage of labour. Cochrane Database Syst Rev 2000;3:CD000007.

20. Roberts CL, Lain SJ, Morris JM. Variation in adherence to recommendations for management of the third stage of labor. Int J Gynecol Obstet 2008;103:172-84.

21. Althabe F, Buekens P, Bergel E, et al. A behavioral intervention to improve obstetrical care. N Engl J Med 2008;358:1929-40.

22. Dildy GA, Paine AR, George NC, Velasco C. Estimating blood loss: can teaching significantly improve visual estimation? Obstet Gynecol 2004;104:601-6.

23. Maslovitz S, Barkai G, Lessing JB, Ziv A, Many A. Improved accuracy of postpartum blood loss estimation as assessed by simulation. Acta Obstet Gynecol Scand 2008;87:929-34.

24. Yasmeen S, Romano PS, Schembri ME, Keyzer J, Gilbert WM. Accuracy of obstetric diagnoses and procedures in hospital discharge data. Am J Obstet Gynecol 2006;194:992-1001.

25. Lain SJ, Roberts CL, Hadfield RM, Bell JC, Morris JM. How accurate is the reporting of obstetric hemorrhage in hospital discharge data? A validation study. Aust N Z J Obstet Gynaecol 2008;48:481-4.

26. Lydon-Rochelle MT, Holt VL, Nelson JC, et al. Accuracy of reporting maternal in-hospital diagnoses and intrapartum procedures in Washington State linked birth records. Paediatr Perinat Epidemiol 2005;19:460-71.